```
     FILE 'USPATFULL, CAPLUS' ENTERED AT 22:44:23 ON 09 JUN 2006
L22         8258 FILE USPATFULL
L23        14764 FILE CAPLUS
     TOTAL FOR ALL FILES
L24        23022 S "IL-10" OR "IL10"
L25          679 FILE USPATFULL
L26          679 FILE USPATFULL
L27           92 FILE USPATFULL
L28           30 FILE CAPLUS
     TOTAL FOR ALL FILES
L29          122 S L24 AND  "DHEA"
L30           11 FILE USPATFULL
L31           27 FILE CAPLUS
     TOTAL FOR ALL FILES
L32           38 S L24 (2S) "DHEA"
L33           11 FILE USPATFULL
L34            5 FILE CAPLUS
     TOTAL FOR ALL FILES
L35           16 S (RHEUMATOID OR (MULTIPLE MYELOMA) OR LYMPHOMA OR (SJOGREN? SY

     FILE 'STNGUIDE' ENTERED AT 22:50:37 ON 09 JUN 2006

     FILE 'USPATFULL' ENTERED AT 22:55:39 ON 09 JUN 2006
L36            9 S L24 AND  "DHEA"/CLM
L37           87 S DHEA/AB AND  "DHEA"/CLM
L38            3 S L37 AND L32

=> save l22-l38
ENTER NAME OR (END):l10623464/l
L# LIST L22-L38 HAS BEEN SAVED AS 'L10623464/L'

=>
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L34 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

AB A review with 22 refs. The progression of **HIV** infection is accompanied by complex alterations in the production of adrenal steroids. Cortisol levels are increased in **HIV** infection whereas those of dehydroepiandrosterone (**DHEA**), a physiol. antagonist of the immunoregulatory activities of cortisol, decrease. The progression of **HIV** infection to **AIDS** is also characterized by a shift from a type 1-to-type 2 cytokine production Thus, defective production of interferon gamma (IFNγ), interleukin (IL)-2, and IL-12 as well as increased production of IL-4, IL-5, IL-6, and **IL-10** are observed in **HIV**-seropos. individuals and are proposed to be in vitro immunol. marker of progression. Cortisol and pharmacol. doses of glucocorticoids (GC) suppress IL-2 and IFNγ production and favor the production of IL-4. Furthermore, GC and IL-4 stimulate the differentiation of B lymphocytes into IgE producing plasma cells, the concentration of which augments in **HIV** infection. Finally, GC induce programmed cell death (PCD) in a variety of different cells, including mature T lymphocytes, and type 2 cytokines were recently proposed to augment the susceptibility of T lymphocytes to PCD. It was suggested that the progressive shift from type 1 to type 2 cytokine production characteristic of **HIV** infection could be at least partially provoked by the increase in the production of cortisol and the reduction of DHEA. This hypothesis is discussed within the scenario of an endrocrinol. imbalance being responsible for **HIV** progression at least partially via increased susceptibility of **HIV** + CD4 lymphocyte to PCD.

ST review **HIV** cortisol cytokine apoptosis lymphocyte

IT **AIDS** (disease)
Apoptosis
B cell (lymphocyte)
Human immunodeficiency virus
T cell (lymphocyte)
(possible role for cortisol/anticortisols imbalance in progression of human immunodeficiency virus)

ACCESSION NUMBER: 1997:552961 CAPLUS
DOCUMENT NUMBER: 127:233160
TITLE: A possible role for the cortisol/anticortisols imbalance in the progression of human immunodeficiency virus
AUTHOR(S): Clerici, Mario; Trabattoni, Daria; Piconi, Stefania; Fusi, Maria Luisa; Ruzzante, Stefania; Clerici, Claudia; Villa, Maria Luisa
CORPORATE SOURCE: Cattedra di Immunologia, Universita degli Studi di Milano, Milan, Italy
SOURCE: Psychoneuroendocrinology (1997), 22(Suppl. 1), S27-S31
CODEN: PSYCDE; ISSN: 0306-4530
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

L34 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

AB Alterations in the production of adrenal steroids and a complex pattern of dysregulation in cytokine profiles accompany the progression of **HIV** infection. Cortisol levels increase in **HIV** infection, while those of dehydroepiandrosterone (**DHEA**), a physiol. antagonist of the immunoregulatory activities of cortisol, decrease. A shift from type-1 to type-2 cytokine production is also detected in most patients during disease progression. This shift is summarized as a defective production of interferon gamma (IFNγ), interleukin-2 (IL), and IL-12 accompanied by increased production of IL-4, IL-5, IL-6, and **IL-10**. IFNγ and IL-2 are suppressed, while the generation of IL-4 is stimulated by cortisol and pharmacol. doses of glucocorticoids (GC). GC and IL-4 stimulate the differentiation of B lymphocytes into IgE-producing plasma cells, the concentration of which augments in **HIV** infection. Finally, GC induces programmed cell death (PCD) in a variety of different cells, including mature T lymphocytes. Because (1) TH1 but not TH2 undergo rapid Fas-mediated PCD upon antigen-stimulation, and (2) TH2 clones preferentially survive in vitro cell cultures, the progressive shift from type-1 to type-2 cytokine production observed in **HIV** infection could be at least partially provoked by the increase in the production of cortisol and the reduction of DHEA. Progression of **HIV** infection to **AIDS** can be controlled by highly active antiretroviral therapy (HAART); HAART drastically reduces **HIV** plasma viremia, but is less effective in immune reconstitution. Addnl. HAART is associated in a sizable portion of patients by complex lipodystrophic phenomena that often involve the endocrine system.

ST **HIV** infection cortisol DHEA cytokine

ACCESSION NUMBER:	2000:898582 CAPLUS
DOCUMENT NUMBER:	135:75587
TITLE:	Immunoendocrinologic abnormalities in human immunodeficiency virus infection
AUTHOR(S):	Clerici, Mario; Galli, Massimo; Bosis, Simona; Gervasoni, Cristina; Moroni, Mauro; Norbiato, Guido
CORPORATE SOURCE:	Cattedra di Immunologia, Universita di Milano, Milan, 20157, Italy
SOURCE:	Annals of the New York Academy of Sciences (2000), 917(Neuroimmunomodulation), 956-961 CODEN: ANYAA9; ISSN: 0077-8923
PUBLISHER:	New York Academy of Sciences
DOCUMENT TYPE:	Journal
LANGUAGE:	English
REFERENCE COUNT:	25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE I

evaluating effects of a pharmaceutical agent (i.e., **DHEA**) or other treatment intervention on progression of a medical condition in a subject, etc.

DETD [0098] The current use of **DHEA** to modulate **IL-10** levels was examined in a randomized, double-blind, and placebo-controlled study conducted as a substudy within a larger multicenter study. See,. . .

DETD [0115] **IL-10** was significantly higher at the time of the last visit in the placebo-treated group as compared to the **DHEA**-treated group (i.e., placebo group 9.06±7.5 vs. **DHEA** group 1.89±1.47 pg/ml, p=0.045). See, Table 3. In addition, the reduction in **IL-10** from baseline to time of last visit was significant within the **DHEA**-treated group (i.e., mean baseline concentration of 9.21±6.75 and mean last visit concentration of 1.89±1.47 pg/ml, p=0.029). See, Table 4.

TABLE 3

Between Group Comparison of cytokine profiles.

	DHEA group	Placebo group	p
IL-1β before treatment (pg/ml)	9.94 ± 8.92	7.22 ± 4.24	0.498
IL-10 before treatment (pg/ml)	9.21 ± 6.75	8.20 ± 6.25	0.577
IL-1β after treatment (pg/ml)	9.20 ± 6.49	9.02 ± 6.83	0.942
IL-10 after treatment (pg/ml)	1.89 ± 1.47	9.06 ± 7.50	0.045

1. Data was presented by mean ± SD (n = 15. . .

DETD [0116]

TABLE 4

Intra-group comparison of cytokine profiles.

	DHEA group	p	Placebo group	p
IL-1β before treatment (pg/ml)	9.94 ± 8.92	0.949	7.22 ± 4.24	0.441
IL-1β after treatment (pg/ml)	9.20 ± 6.49		9.02 ± 6.83	
IL-10 before treatment (pg/ml)	9.21 ± 6.75	0.029	8.20 ± 6.25	0.519
IL-10 after treatment (pg/ml)	1.89 ± 1.47		9.06 ± 7.50	

1. Data was presented by mean ± SD (n = 15 for each. . .

DETD [0121] However, in this double-blind study conducted to evaluate the effects of **DHEA** on cytokine profiles in lupus disease, the inventors found significant reduction of **IL-10** in patients' blood after **DHEA** treatment for 24 weeks.

DETD [0123] The current finding of significant **IL-10** suppression by treatment of 200-mg/day dosages of **DHEA** daily for 24 weeks in adult Chinese female patients with mild to moderate SLE is, thus, thought to explain why **DHEA** offers meaningful benefit especially to steroid-dependent lupus patients.

CLM What is claimed is:

1. A method of modulating a level of **IL-10** in a subject, the method comprising: a) selecting a subject in need of a modulated **IL-10** level; and, b) administering an

amount of **DHEA** to the subject, wherein the amount is effective to modulate the level of **IL-10** in the subject.

3. A method of modulating a level of **IL-10** in a subject, the method comprising: a) administering an amount of **DHEA** to the subject, wherein the amount is effective to modulate the level of **IL-10** in the subject; and, b) measuring the level of **IL-10** in the subject.

26. The method of claim 1, 2, or 3, wherein administering comprises administration of **DHEA** and one or more of: a glucocorticoid, a monoclonal antibody specific for **IL-10** or a fragment of **IL-10**, an immunosuppressant, an anti-malarial drug, an alkylating agent, or a chemotherapeutic agent.

27. The method of claim 2 or 3, wherein measuring the level of **IL-10** in the subject comprises measuring a basal level of **IL-10** in the subject prior to administering the amount of **DHEA**.

28. The method of claim 27, wherein measuring the level of **IL-10** in the subject additionally comprises measuring a level of **IL-10** in the subject after administering the amount of **DHEA**.

29. A method of prophylactically or therapeutically treating one or more medical conditions in a subject, the method comprising modulating a level of **IL-10** by: a) administering a first amount of **DHEA** to the subject; b) measuring the level of **IL-10** in the subject; and, c) administering at least a second amount of **DHEA** to the subject, wherein the second amount is determined based upon the level of **IL-10** measured in step (b).

L38 ANSWER 3 OF 3 USPATFULL on STN

ACCESSION NUMBER:	1998:127915 USPATFULL
TITLE:	Vaccine compositions and method for induction of mucosal immune response via systemic vaccination
INVENTOR(S):	Daynes, Raymond A., Park City, UT, United States Araneo, Barbara A., Salt Lake City, UT, United States
PATENT ASSIGNEE(S):	University of Utah Research Foundation, Salt Lake City, UT, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5824313		19981020
APPLICATION INFO.:	US 1995-480567		19950607 (8)

RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1993-123844, filed on 9 Sep 1993, now patented, Pat. No. US 5518725 which is a continuation-in-part of Ser. No. US 1993-13972, filed on 4 Feb 1993, now abandoned And Ser. No. US 1991-779499, filed on 18 Oct 1991, now abandoned which is a continuation-in-part of Ser. No. US 1989-412270, filed on 25 Sep 1989, now patented, Pat. No. US 5540919
DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Housel, James C.
ASSISTANT EXAMINER:	Swartz, Rodney P.
LEGAL REPRESENTATIVE:	Venable, Baetjer, Howard & Civiletti, LLP
NUMBER OF CLAIMS:	61
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	66 Drawing Figure(s); 24 Drawing Page(s)
LINE COUNT:	2188

L39 ANSWER 22 OF 24 USPATFULL on STN

SUMM . . . crosslinking Fe receptors present on the cell surfaces of antigen presenting cells. Other embodiments relate to methods of increasing the **levels** of **IL-10** in an individual in **need** thereof. Still further embodiments relate to methods of stimulating peripheral tolerance and/or bystander suppression in an individual in need thereof.. . .

SUMM . . . embodiment of the present invention is a method of reducing disease symptoms in an individual comprising identifying an individual in **need** of an increased **level** of **IL-10** and increasing the **level** of **IL-10** in said individual by administering a composition comprising an immunoglobulin or portion thereof linked to an antigen, wherein said immunoglobulin. . .

SUMM . . . embodiment of the present invention is a method of reducing disease symptoms in an individual comprising identifying an individual in **need** of an increased **level** of **IL-10** and in **need** of stimulation of peripheral tolerance and increasing the **level** of **IL-10** and stimulating peripheral tolerance in said individual by administering a composition comprising an immunoglobulin or portion thereof linked to an. . .

CLM What is claimed is:

34. A method of reducing disease symptoms in an individual comprising: identifying an individual in **need** of an increased **level** of **IL-10**; and increasing the **level** of **IL-10** in said individual by administering a composition comprising an immunoglobulin or portion thereof linked to an antigen, wherein said immunoglobulin. . .

49. A method of reducing disease symptoms in an individual comprising: identifying an individual in **need** of an increased **level** of **IL-10** and in **need** of stimulation of peripheral tolerance; and increasing the **level** of **IL-10** and stimulating peripheral tolerance in said individual by administering a composition comprising an immunoglobulin or portion thereof linked to an. . .

AN 2002:67349 USPATFULL|

PI US 2002038002 A1 20020328|

L39 ANSWER 11 OF 24 USPATFULL on STN
DETD . . . rodents, the mutual relationship of which is not yet finally defined. The so-called Tr1 and Th3 cells mediate bystander suppression--without **need** for direct cell contact--by the secretion of high **levels** of **IL-10** and TGF-β, respectively (Groux, H. et al., Nature 389:737-742 (1997); Fukaura, H. et al., J. Clin. Invest. 98:70-77 (1996)). The. . .
AN 2005:117745 USPATFULL
PI US 2005101012 A1 20050512

L39 ANSWER 12 OF 24 USPATFULL on STN
DETD . . . achieve a therapeutic effect. As discussed above, a dose of IFNτ on the order of greater than 5+10.sup.8 Units is **needed** to achieve a measurable increase in a patient's blood **IL-10 level**. The same increase in IL-10 level can be achieved with a lower dose of IFNτ when the IFNτ is administered. . .
DETD . . . of IFNτ and a second part comprised of components required to monitor a biomarker of IFNτ, such as the components **needed** to analyze blood **IL-10 levels**
.
AN 2005:98563 USPATFULL
PI US 2005084478 A1 20050421

L39 ANSWER 13 OF 24 USPATFULL on STN